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BORDEN'S REVIEW *of* NUTRITION RESEARCH

DIETARY CAUSES OF CONGENITAL ABNORMALITIES

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DIETARY CAUSES OF CONGENITAL ABNORMALITIES

Nutritional inadequacy during pregnancy presents a two-fold problem: first, the effect on the mother at whose expense the embryo is nourished; and second, the influence on the course of development of the embryo or fetus. It has been generally assumed that where maternal undernutrition exists, the developing embryo or fetus will obtain the necessary nutriment by drawing upon the maternal tissue stores, so that the primary effect of dietary inadequacy will be evidensed in the mother. This assumption has been generally accepted and is fairly well established. Recent animal experiments reveal, however, that maternal protection of the developing embryo is not always adequate and that in certain borderline cases, maternal dietary deficiency may result in the birth of malformed young. Further, it has been found possible to induce congenital abnormalities at will, independent of genetic factors, by maintaining the mother on a special deficient diet. These experiments may have an important bearing on problems of prenatal nutrition and certain congenital deformities in man.

Early Experimental Observations:

The earliest indications that deficient maternal diets may exert adverse effects upon the offspring came from the field of animal husbandry. The classical experiments of Hart and coworkers (1, 2, 3) during the period from 1911 to 1924 suggested an important relationship between nutrition and reproduction. They noted that certain grain rations fed to cattle frequently led to reproductive failure or to the birth of premature, weak or dead offspring. In later work, this effect was traced to a lack of vitamin A in the rations used.

In 1921, not many years after the discovery of the fat-soluble vitamins A and D, a group of British investigators (4) made the interesting observation that sows fed a diet deficient in vitamin A produced pigs in which some limbs were completely missing. Ten years later, Converse and Meigs (5) observed congenital blindness in the offspring of cows fed over-ripe timothy hay low in vitamin A. Other investigators reported blindness, deformities and paralysis in pigs and cattle which were attributed to nutritional deficiencies in the maternal diet (6, 7).

Nutrition vs. Heredity: Following these and numerous other observations on abnormalities in offspring of inadequately fed mothers,

attempts were made to rule out hereditary factors and to trace the occurrence of the abnormalities unequivocally to nutritional faults. Pioneer work in this field was done by Hale at the Agricultural Experiment Station in Texas.

In connection with an investigation on the effects of vitamin A deficiency on swine, Hale (8) fed a gilt a vitamin A free ration for more than five months before breeding and for one month following breeding. This pig subsequently produced a litter of eleven pigs all of which were born without eyeballs. Since an anomaly of this nature had never been observed in the herd, it was concluded that the abnormality was in some way related to the maternal deficiency of vitamin A. Further experiments were immediately initiated to determine the relation between vitamin A and embryonic eye development (9, 10). Young female pigs were depleted to a very low level of vitamin A before breeding and for the first thirty days thereafter (the period during which the eye develops in the pig embryo). One of the gilts failed to exhibit normal estrus and was, therefore, not bred, while another failed to produce a litter following breeding, due to early resorption of the fetuses. Two other gilts on the vitamin A deficient regimen succeeded in producing litters. The first of these latter gilts had been given a single dose of cod liver oil two weeks before conception because the severity of the vitamin A deficiency was such as to prevent the animal from being able to stand; the other went through the entire period without vitamin A. The litter produced by this latter animal consisted of ten pigs all without eyeballs—an exact duplicate of the first experiment. The animal which had received a single emergency dose of cod liver oil before breeding produced fourteen pigs with various combinations of eye defects; some with no eyes; some with one eye; some with one large and one small eye; but all blind. In addition, various other defects were noted in both litters. These included accessory ears, subcutaneous cysts, hare lip and misplaced kidneys. Control animals receiving the vitamin A deficient diet plus regular supplements of cod liver oil all farrowed normal young.

Definite and complete genetic tests were conducted to determine whether or not an unsuspected hereditary factor was responsible for the eye anomalies and other defects. Close matings on adequate diets such as blind brother and blind sister, normal mother and blind son, and so on, produced only normal pigs. These experiments pointed clearly, therefore, to the fact that these congenital deformities were the direct result of maternal deficiency in vitamin A. Hale

postulated, on the basis of these studies, that various eye defects and weaknesses in the human race may have a similar origin in maternal deficiency.* It is pointed out, however, that human mothers rarely have a dietary as extreme and exaggerated in lack of vitamin A as that supplied to experimental animals. Nevertheless, in view of the variety of defects encountered in the animal given a single dose of cod liver oil, Hale suggests the possibility that there are various degrees in which a lack of vitamin A can affect eye development.

Malformations in Poultry: With poultry, as with pigs and cattle, deficiencies in maternal diets were soon recognized as an important cause of malformation in the offspring. The field was broadened, moreover, with the realization that lack of nutrients other than vitamin A was productive of disturbances and abnormalities in embryonic development.

In 1935, Byerly and associates (11) at the Beltsville Research Center in Maryland reported the occurrence of a hitherto undescribed disease causing the death of many chick embryos during the third week of incubation of eggs produced by chickens on experimental diets. The diseased embryos were characterized by abnormal shortness of the bones, particularly of the skull and legs. Occasionally, a few of the malformed embryos succeeded in hatching. These chicks behaved abnormally, presenting an appearance very similar to that caused by polyneuritis. An adequate diet was successful in restoring normal behavior to a few of these chicks in the course of ten weeks. Tests showed the experimental diet of the hens to be very low in thiamin and riboflavin, while the eggs which they produced were very deficient in thiamin and vitamin E, but not in riboflavin. Supplements of thiamin and riboflavin were without effect, but wheat germ, a rich source of vitamin E, succeeded in preventing the disease. Whey and dried liver supplements were also successful. Exposure of the hens to direct sunlight and access to green range markedly enhanced the preventive properties of the supplements. The specific preventive factor or factors in the supplements were not determined.

About two years later, Lyons and Insko (12) during their investigation of "slipped tendon" (perosis) in chicks resulting from certain mineral deficiencies, reported the occurrence of malformed embryos in eggs produced by hens receiving a diet deficient in man-

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* The birth of a healthy human infant without eyeballs has recently been reported in the daily press. Since this case has not yet been reported in medical journals, no conclusions as to the cause of this defect, nutritional or otherwise, can be drawn.

ganese.* The embryos showed very short legs and wings and "parrot beak." The majority of these chicks failed to hatch. The few which hatched, did not have slipped tendon despite the bone malformations. The disease, termed *chondrodystrophy,* was prevented by the injection of 0.03 mg. of manganese directly into the albumen of such eggs just prior to incubation. Shortly following this work Lepkovsky *et al* (13) reported that riboflavin deficiency in the diet of the breeding hen caused degenerative changes in the tissues, reduced size of the embryo and frequently death of the embryo.

Recent Experimental Studies:

Since the presentation of Hale's report (9) on eye defects in pigs born of vitamin A deficient sows, Cannon (14) has reported failure to induce congenital anomalies in the offspring of vitamin A deficient rats. This may possibly be due to species differences or other causes. However, in 1941 Andersen (15) suggested, on the basis of limited experiments with rats, that the lack of vitamin A in maternal diets was a probable factor in the development of congenital hernia of the diaphragm. Hereditary influences were not excluded in her experiments, however, nor was further confirmatory evidence reported.

Experimentally Induced Malformations: Beyond the observations originating in animal and poultry husbandry, and the brief, scattered reports of isolated laboratory experiments, no precise scientific knowledge existed until recently as to the validity of the assumption that congenital anomalies may be brought about as a result of deficiencies in the maternal diet. In June of this year, however, a report was published describing a series of congenital deformities in experimental animals which was directly and unequivocally traced to a specific deficiency in the maternal diet. Moreover, these deformities could be produced or prevented at will by regulating the maternal diets.

This interesting report culminated a series of carefully planned and controlled experiments begun about five years previously by a group of scientists at the Children's Hospital Research Foundation in Cincinnati and the Department of Pediatrics of the University of Cincinnati. In 1940, Warkany and Nelson (16) of this group, published a brief note to the effect that rats fed the standard Steenbock and Black rachitogenic diet, supplemented with vitamin D to prevent rickets (diet 1), gave birth to young of which more than

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* See *Borden's Review of Nutrition Research,* January, 1944 on "Manganese."

one-third presented marked skeletal defects. These anatomical defects were studied and described in complete detail in later reports (17, 18, 21). The defects consisted chiefly of malformations of the extremities, of the jaw and of the ribs. Bones of the forelegs and hindlegs were foreshortened or, in some cases, completely missing. The bones of toes were formed abnormally or were fused together forming forefeet and hindfeet with less than the usual number of digits. The mandible, in many cases, was so markedly shortened that the tongue protruded from the jaw. By means of an ingenious method devised by Schultze and Dawson the specimens were "cleared" (the soft tissue rendered transparent, while the bones, stained bright red, stand out clearly under transillumination) so that skeletal abnormalities other than those externally apparent could be detected. Extreme malformations of the ribs were evidenced in the cleared specimens—the ribs were shortened, twisted, fused together, or absent. The exact nature of the other externally visible deformities was also studied by the clearing method. The deformed animals are usually born dead or die soon after birth, thereby precluding study of the further development of the malformations. In the case of one abnormal animal which survived and was raised to adulthood on an adequate diet, however, a roentgenogram of the chest revealed the fusion of the ribs to be a permanent malformation. Later work brought to light another abnormality, cleft palate, which appeared to be a part of the regular pattern of malformations caused by the deficient diet, although occurring with less frequency than the other defects. Warkany, Nelson and Schraffenberger (22) reported in detail on the special type of cleft palate resulting from maternal nutritional deficiency and emphasized clearly the fact that this deformity can result from environmental (in this case, nutritional) modification of fetal growth as well as from hereditary causes.*

Histologic studies were made of the various bone tissues in the hope of determining the mechanism involved in the development of the abnormalities. Studies of a leg bone revealed a persistence of cartilage in areas where normally ossification should have taken place. Aside from the delay in ossification, the center of calcification which

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*As yet, little information of practical significance with respect to the occurrence of congenital deformities in children can be drawn from this work, although the implications are obvious and merit further study. With respect to cleft palate, in particular, however, the authors state: "The experiments reported . . . should encourage attempts to modify facial cleft formations in strains, and also in families, in which such malformations are hereditary. Whether modification by nutritional measures will be possible or not, the experimental production of phenocopies (duplication of the effects of a mutation by an environmental modification) of cleft palates proves that it is not justifiable to assume *a priori* that all cleft palates in human beings are hereditary . . . It is more likely that the clinical material encountered is a conglomeration of hereditary malformations and phenocopies."

should have developed in the center of the bone structure, actually was found near the posterior surface of the bone. This led to abnormal structural arrangements with consequent weakness and further distortion of the developing bone.

In general, the abnormalities were confined to the skeletal structure, although severe malformations of the soft tissues occasionally were found. These consisted of deformities of the eyes, gastroschisis (congenital malformation in which the abdomen remains open) and hemiatrophy (wasting away of one lateral half of an organ).

The authors made the discovery that the malformations could be completely prevented by the simple expedient of incorporating two per cent of dried pig liver in the diet of the mother (16). Further, when the same pair of animals were bred alternately on the deficient diet and on the same diet containing pig liver in addition, abnormal and normal offspring could be obtained alternately. These circumstances led the authors to conclude that they were dealing with a nutritional phenomenon:

> "A nutritional factor absent in diet I but contained in large amounts in liver is apparently necessary for the normal intrauterine development of the fetus. Without this factor certain bones show arrest of development and malformations at the time of birth."

Quest for Nutritional Factor: By 1942, the group in Cincinnati had determined beyond doubt that the characteristic pattern of skeletal deformities observed in their animals was the direct result of a deficiency in the diet of the mother. The deformities could be produced at will and, more significant, could be prevented at will by the addition of dried pig liver. There were two possible approaches to the discovery of the factor or factors responsible for this remarkable protection. The first of these involved a careful study of the exact deficiencies of the Steenbock-Black rachitogenic diet.

This was not a highly purified synthetic diet, but was one of the older types which supplied carbohydrate, protein, minerals and some fat and vitamins in the form of yellow corn meal, wheat gluten, calcium carbonate and sodium chloride. The obvious deficiency was, of course, vitamin D, but this had been corrected by periodic supplements of viosterol. The diet was known to be low in iodine, mothers and offspring alike showing hypertrophy and hyperplasia of the thyroid gland. The possibility was considered that the maternal goiter might influence the embryonic development of the young. This was readily disproved, however, when supplements of iodized salt failed to prevent the abnormalities (20). In view of the reports on chondro-

dystrophy in chicks resulting from manganese deficiency in the diet of the hen (12), supplements of this mineral were given but proved unsuccessful. Similar failures were obtained with supplements of the animal protein, casein, of alfalfa meal (a good source of vitamins A, E, K, pantothenic acid and pyridoxine), of cod liver oil and of wheat germ oil.

Riboflavin, the Preventive Factor: The one remaining avenue of investigation was to follow up the clue afforded by the marked protective power of pig liver. Since liver contains large amounts of many of the B-complex vitamins, a series of long, intensive experiments was initiated to test several crystalline substances of this group including thiamin, riboflavin, niacin, pyridoxine and calcium pantothenate (24).

> The problem was complicated by the fact that the stores of the maternal tissues act as "buffers" which prevent deprivation of the developing embryo as long as possible. As the authors point out, "it was assumed until recently that these maternal stores either protect the offspring completely, thus resulting in the delivery of normal young, or that in case of extreme dietary deficiency the embryos die *in utero*. Although there is some truth in this 'all or none' theory, it is not entirely correct. *Between the two there exists a narrow range in which maternal nutritional deficiency may result in arrest of the embryos' development without causing death. In this case congenitally deformed offspring may be the result.*"

Because the phenomenon of malformed young was essentially the manifestation of a borderline deficiency not more than one-third of each of the litters could be expected to be abnormal. This entailed the rearing of large numbers of young before it was possible to credit a test supplement with preventive properties. The basic deficiency diet (diet 1) was supplemented with each of the five B vitamins, singly and in various combinations. When all five were added at once, complete protection of the offspring was obtained. This

TABLE 1. *The effect of vitamin supplementation of diet I on the appearance of congenital malformations.*

MOTHERS FED DIET I SUPPLEMENTED BY:	OFFSPRING		
	Total	Normal	Abnormal
Riboflavin + thiamine + niacin + pyridoxine + Ca pantothenate	371	371	0
Thiamine + niacin + pyridoxine + Ca pantothenate	51	27	24
Pyridoxine + Ca pantothenate	67	47	20
Thiamine	152	125	27
Niacin	180	161	19
Riboflavin	319	319	0

meant the investigation was at last on the right track. The vitamins were then tried in groups of four, two and singly (Table 1).

The results were unmistakable. One, and only one, of the five B vitamins could prevent the abnormalities—*riboflavin*.

Additional confirmation of the preventive power of riboflavin was obtained when diet 1 was replaced by purified components (sucrose, vitamin-free casein, vegetable oil, salt mixture, vitamins A and D and the crystalline B vitamins under investigation) (23). Again, the preventive factor was found to be riboflavin. A modification of this supplemented, purified diet (containing vitamins E and K, in addition) without riboflavin (diet Z) was used in repetitions of these experiments. The females were kept on diet Z for some weeks prior to breeding thereby greatly increasing the yield of abnormal offspring. In the case of some females who became so affected by riboflavin deficiency as to exhibit loss of weight, absence of estrus cycles and inability to reproduce, a system of "controlled restoration" was adopted. This meant the animals were given small amounts of riboflavin just sufficient to restore weight and reproductive functions. In these "restored" animals, the incidence of abnormal young rose to more than 50 per cent.

Crucial Embryologic Period: One of the most interesting aspects of the studies conducted by the Cincinnati investigators involved the determination of the actual period in embryonic development when the lack of riboflavin resulted in abnormal tissue differentiation.

In rats, where the gestation period is twenty-one days, the formation of the "fingers," for example, is such that the number of digits becomes recognizable on the fourteenth day of embryonic life. From this it may be concluded that the development of an abnormal number of digits must occur before the fourteenth day. The question is: precisely when, before the fourteenth day, is this malformation determined? When is the need for riboflavin at the crucial point?

The answer to this question was ingeniously determined by the use of alternating diets. Early in their experiments, it had been noticed that abnormal offspring could be prevented regularly when the change was made to a liver-containing diet before the next mating. With this fact as a basis, a series of experiments was planned in which the dietary change was made *on different days after mating* (20). By this procedure it was hoped to be able to ascertain exactly the day of gestation before which the offspring could be protected by adding liver to the mother's diet and after which such protection would be impossible.

Accordingly, the experiments were planned so that a rat which had previously had an abnormal litter on the deficient diet was again mated and given the protective liver supplement one day after mating. Another such rat was mated and given the supplement not until the second day after mating. The same was done with still other rats until such experiments with alternating diets were performed for each of the twenty-one days after conception.

The results showed that the mother rat could produce normal young if the liver supplement was given (and maintained) at any time up to the thirteenth day of gestation. In other words, a rat on a deficient diet for as long as the first twelve days could still produce normal young. By the thirteenth day, however, the lack of riboflavin became crucial—if liver was then supplied, the young were protected; if not, abnormal young resulted. Adding the liver on the fourteenth day, or any day thereafter, failed to protect the young from developing abnormally.

It was thus determined that, in the case of rats, the ability of riboflavin to influence normal tissue differentiation is exerted in the relatively brief period between the 13th and 14th days of gestation—or well past the halfway period. In this connection, it may be well to point out that developmental stages vary with species. In the rat, the embryonic period, the period of differentiation, is relatively long and the fetal period, the period of growth, very short. In humans, however, differentiation begins early, and is usually fairly complete by the end of the first third of pregnancy, while the remaining time is devoted to growth. If results of these experiments were to apply to humans, therefore, the critical period when prenatal riboflavin nutrition would be most important would be some time during the first three months of fetal life.

The implications of these dramatic experiments with respect to maternal human nutrition are tremendous. While the occurrence of congenital human abnormalities is reassuringly rare, the question of degree remains to be considered. No longer is there the certainty that the developing infant will be protected at all costs by drawing on the mother's stores. The borderline riboflavin deficiency in animals wherein the mother is barely able to conceive and wherein the fetus has just enough riboflavin for growth, but not for differentiation, may very likely have an analogue in human reproduction. It is possible that the animal experiments described may afford some insight into the intricacies of the problems involved in human maternal nutrition and reproduction.

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NEWS DIGEST

VITAMIN C FROM BEETS—A new process makes it possible now to obtain vitamin C from beet pulp following sugar extraction. The vitamin can be readily prepared by simple methods from galacturonic acid present in the dried beet pulp. The dry pulp contains about 30 per cent of galacturonic acid and since an estimated 7,000,000 tons of beet pulp are available annually, a practically unlimited source of raw material for vitamin C manufacture would be afforded. It is hoped that the new procedure will augment normal supplies of this vitamin from fruits and vegetables. Demands for vitamin C have been increased by the fortification of certain foods for military use, and also because of medical usage to accelerate the healing of wounds. Industrial development of the new process is expected. This may come by processing beet pulp or by using the same chemical methods on citrus wastes which are also high in pectic substances. Apparently, any pectic substance can be satisfactorily used as a starting material. *Food Ind.*, July, 1944.

pH INDICATES FOOD SPOILAGE—Spoilage of fish can be determined instantly and with quantitative accuracy by merely touching a sample of the fish to the glass electrodes of a pH apparatus, according to a recent report from Canada. Spoilage, it is pointed out, occurs almost entirely through bacterial action at the surface of the fish. The surface measurement of the end products of such bacterial action, therefore, provides the most satisfactory index of spoilage. It has been found that as these end products accumulate, indicating progressive spoilage of the fish, the pH value of the fish surface increases (the surface becomes more alkaline in reaction). Contact with the glass electrodes permits precise determination of the pH and, therefore, of the degree of spoilage. The dial on the pH instrument can be marked for ready reading, "fresh," "spoiling," "spoiled," conditions which correspond to a range of pH values from 6.0 to 8.0. *Food Field Reporter*, July 24, 1944.

DRY CHEESE MIX — A newly perfected dry cheese mix which can be stored safely for 12 months without refrigeration has been developed for shipment to protein-starved Europe after the war. The mix contains dehydrated cheddar cheese, non-fat milk solids, wheat and soy flour and salt. It is easily reconstituted with cold water and may be prepared as a soup, spread or sauce, depending upon the amount of water added. Because it is intended for use in many different countries whose food habits differ, spices and flavorings have been omitted, leaving the mix quite bland and readily adaptable to local tastes. The product is made in the form of one-pound compressed bricks and packaged in special moisture-proof containers. Directions for reconstituting the mix are printed on the wrapper in 16 languages. *Food Ind.*, July, 1944.

CEREALS IN ARMY RATIONS—A new development by the Quartermaster Corps will put America's favorite breakfast cereals in the food kit of every soldier fighting overseas. The cereals in granulated form have been compressed with powdered milk, sugar, and salt into discs only 2 11/16 inches in diameter to fit into the round "C" ration can used in active combat zones. The discs, less than an inch thick, can be eaten dry or crumbled into a mess kit and softened with hot or cold water. For

variety they have been prepared of a flaked wheat cereal, a whole wheat product, and a cornflake-grape-nuts type, each containing about two ounces of cereal. *Food Field Rpt.,* July 10 (1944).

PYRIDOXINE IN MILK – Interesting data have recently been reported with respect to the pyridoxine (vitamin B_6) content of cow's milk. Samples of fresh, pasteurized, evaporated and powdered milks from various states were collected and assayed for the vitamin. An average value of 0.67 mg. pyridoxine per liter (1.06 qts.) of milk is reported, with no significant losses in the preparation of pasteurized, evaporated or dried milk. On a dry basis milk compares favorably with cereals in pyridoxine content and contains about one-hal to one-fourth as much as most meat *J. Nutr.* 27, 415 (1944).

BROCCOLI – Broccoli leaves, drie and ground into meal, have been foun to be an excellent addition to chicke feed mash according to tests conducte at the Eastern Regional Laboratory o the U. S. Department of Agriculture. Leaf meals from five kinds of plant were prepared and tested. These in cluded broccoli, lima bean, carrot, tur nip and pea. Of these, broccoli prove to be the best, showing high vitamin content and a percentage of protein substantially higher than that of alfalfa meal. Flavor of the meat of broccoli-fe chickens is said to be definitely im proved. *Sci. 100,* Suppl. Sept. 1 (1944).